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Difference in the Respiratory Variation Between Pulmonary Venous and Mitral Inflow Doppler Velocities in Patients With Constrictive Pericarditis With and Without Atrial Fibrillation

Tomotsugu Tabata, MD, PHD, Samer S. Kabbani, MD, R. Daniel Murray, PHD, James D. Thomas, MD, FACC, Ibrahim Abdalla, MD, Allan L. Klein, MD, FACC
Cleveland, Ohio

OBJECTIVES The goal of this study was to evaluate the difference in the respiratory change from expiration to inspiration (%E) between pulsed Doppler mitral inflow (MV) and pulmonary venous flow (PV) velocities in patients with constrictive pericarditis (CP) and to describe the influence of atrial fibrillation (AF).

BACKGROUND The difference in %E between MV and PV velocities as well as the influence of AF on %E has not been well described.

METHODS Pulsed-wave Doppler transesophageal echocardiography (TEE) was performed with respiratory monitoring in 31 patients with CP and sinus rhythm (SR) and in 10 patients with CP and AF. The MV early (E) and late diastolic (A) velocities and their velocity time integral (VTI) as well as PV systolic (S) and diastolic (D) velocities and their VTI were measured.

RESULTS Regardless of the cardiac rhythm: 1) The MV-E velocity and E-VTI as well as PV-D velocity and D-VTI significantly decreased from expiration to inspiration; 2) the %E in PV-D velocity (27% in SR and 35% in AF) and D-VTI (38% in SR and 45% in AF) was significantly greater than that in MV-E velocity (18% in SR and 15% in AF) and E-VTI (21% in SR and 19% in AF), respectively; 3) the PV S/D and S/D-VTI significantly increased from expiration to inspiration.

CONCLUSIONS A significant respiratory variation was observed in both MV and PV velocities in CP, not only in patients with SR but also in those with AF. Moreover, the %E was greater in the PV velocities than it was in the MV velocities. Evaluation of the %E in the PV velocities using TEE can be a sensitive diagnostic strategy for evaluation of patients with CP, even in patients with AF. (J Am Coll Cardiol 2001;37:1936–42) © 2001 by the American College of Cardiology

The hemodynamic characteristics of constrictive pericarditis (CP) have been well established and include markedly elevated atrial and ventricular pressures and early diastolic "dip and plateau" patterns in the ventricular pressure curves (1). However, the difficulty in diagnosing CP is that there is a similar hemodynamic profile in patients with restrictive cardiomyopathy (2). The sensitivity of the conventional two-dimensional echocardiographic criteria for diagnosing CP varies from 62% to 93% (3,4).

The usefulness of the respiratory variation in the Doppler flow velocities for differentiating CP and restrictive cardiomyopathy has been reported by several investigators (5–7). A marked respiratory variation in Doppler flow velocities in patients with CP was first described by Hatle et al. (5) using transthoracic Doppler echocardiographic interrogation of atrioventricular inflow and ventricular outflow. We have also reported a marked respiratory variation in patients with CP in pulmonary venous (PV) flow velocities by transesophageal echocardiography (TEE) (7). However, the difference in the respiratory variation between the mitral inflow (MV) and PV flow velocities has not been clearly described. Also, the effect of atrial fibrillation (AF) on the respiratory variation in patients with CP has not been well evaluated.

Therefore, the purposes of the study were: 1) to evaluate the difference in the respiratory variation between pulsed-wave Doppler MV and PV flow velocities in patients with CP, and 2) to describe the influence of AF on the respiratory variation.

METHODS

Patient population. Forty-one patients with predominantly right heart failure and clinically suspected diastolic dysfunction were diagnosed as having CP on the basis of a constellation of diagnostic tests, including cardiac catheterization, transthoracic echocardiography, computed tomography, magnetic resonance imaging and surgical findings. Constrictive pericarditis was defined as a disease with a scarred or fused pericardium with reduced atrial and ventricular distensibility and producing significant right heart

From the Cardiovascular Imaging Center, Department of Cardiology, The Cleveland Clinic Foundation, Cleveland, Ohio.

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Abbreviations and Acronyms

A	=	mitral inflow late filling wave
AF	=	atrial fibrillation
ANOVA	=	analysis of variance
CP	=	constrictive pericarditis
D	=	pulmonary venous diastolic wave
E	=	mitral inflow early filling wave
MV	=	mitral inflow
PV	=	pulmonary venous
S	=	pulmonary venous systolic wave
TEE	=	transesophageal echocardiography
VTI	=	velocity time integral
%E	=	respiratory change from expiration to inspiration

failure (8). We excluded the patients with restrictive cardiomyopathy, such as advanced cardiac amyloidosis whose primary abnormality was impaired ventricular compliance caused by abnormal changes in myocardium. The patients with CP who showed mixed constrictive and restrictive physiology and the patients with effusive CP were also excluded from the study (9). The patients with chronic obstructive pulmonary disease were excluded because of confounding effects on respiratory variation of Doppler flows. There were 36 men and five women with a mean age of 58 ± 12 years (range 30 to 79 years). The cardiac rhythm was normal sinus rhythm in 31 (76%) and AF in 10 (24%) of the 41 patients. Twenty-one (68%) of 31 patients with sinus rhythm and seven (70%) of 10 with AF were in New York Heart Association functional class III or IV. The etiology of CP was secondary to previous cardiac surgery in 14 patients (34%), irradiation in 4 patients (10%) and idiopathic in 21 patients (51%). The two other patients had etiologies of collagen vascular disease and pulmonary tuberculosis. Cardiac catheterization and magnetic resonance imaging were performed in 37 (90%) and 33 (80%) of the 41 patients, respectively. Twenty-six (63%) of 41 patients underwent pericardiectomy (mean 51 ± 78 days; range 1 approximately 370 days). The diagnosis of CP was verified by cardiac catheterization in five patients, by magnetic resonance imaging in two patients and by both in eight of 15 patients who were treated medically.

Six patients with AF without any findings of CP or restrictive cardiomyopathy undergoing TEE served as a control group. There were five men and one woman with a mean age of 64 ± 17 years (range 34 to 77 years). Three of them had a history of cardiac surgery, one had a dilated cardiomyopathy (nonrestrictive) and two were without significant cardiac disease. None of patients had evidence of significant mitral insufficiency.

Hemodynamic evaluation. Left ventricular end-diastolic pressure was obtained by pig tail catheter. The right ventricular end-diastolic pressure and pulmonary capillary wedge pressure were obtained by Swan-Ganz catheter. The pulmonary capillary wedge pressure was used for an estimation of the left atrial pressure (10).

Transthoracic echocardiography. Transthoracic echocardiography was performed immediately before TEE using 3.5 MHz transducer attached to commercially available equipment (Sonos 1500 or 2500, Hewlett-Packard Co., Andover, Massachusetts, or Acuson Computed Sonography model 128, Sequoia, Acuson Inc., Mountain View, California). From the parasternal long-axis view, left ventricular end-diastolic and end-systolic dimensions and left atrial dimension were obtained by M-mode method. The left ventricular ejection fraction was calculated according to the method of Quinones et al. (11).

TEE. Transesophageal echocardiography was performed according to established techniques (12) using 5 MHz phased-array biplane and multiplane transducers. From the pulsed Doppler velocity profiles, the peak MV early (E) and late (A) diastolic velocities and their ratio (E/A) as well as the velocity time integrals (E-VTI and A-VTI) were measured. The deceleration time was calculated from the MV-E velocity extrapolated to its baseline. The peak PV systolic (S) and early diastolic (D) velocities and their ratio (S/D), as well as the velocity time integrals (S-VTI and D-VTI), were also measured. Both the Doppler velocity profiles were recorded on the strip chart with a paper speed of 50 or 100 cm/s. Velocity time integrals were measured off-line by digitizing the darkest portion of the Doppler tracings. A nasal respirometer (Interspec, Waterstown, New York, or Acuson Corp., Mountain View, California) was used simultaneously to record the phase of inspiration and expiration (13). The pulsed Doppler TEE recording with respirometry of the MV and PV flows added 5 to 10 min to the clinical transesophageal echocardiography study.

Respiratory measurements. Mean value of Doppler velocities was calculated as averaged peak velocities at the onset of inspiration and expiration for at least three to six respiratory cycles. In the presence of AF, six respiratory cycles were used (12 cardiac cycles). The respiratory variation (%E) in the Doppler velocities and the velocity time integrals from expiration to inspiration were calculated by the formula: %E = (expiration − inspiration)/expiration × 100 (%), according to the previous methods (14).

Statistical analysis. The data were expressed as a mean value ± SD. A paired *t* test was used for the comparison between the values during expiration and inspiration. Analysis of variance (ANOVA) was performed to compare the difference in the %E between the parameters within the groups. Significant results from ANOVA were further analyzed by Bonferroni test to identify significant differences. An unpaired *t* test was used for the comparison of the difference in the %E of each variable between the patients with CP and sinus rhythm and those with CP and AF. A p value <0.05 was considered statistically significant.

RESULTS

Clinical characteristics of the subjects. Table 1 shows the clinical features of all patients with CP subdivided by

Table 1. Clinical Characteristics of the Subjects

	SR-CP (n = 31)	AF-CP (n = 10)	AF Without CP (n = 6)
Age (yr)	57 ± 13	61 ± 11	64 ± 17
Men/Women (n)	28/3	8/2	5/1
LAD (cm)	4.3 ± 0.5	5.5 ± 1.0*	4.7 ± 0.5
LVDd (cm)	4.8 ± 0.6	4.7 ± 0.9	5.5 ± 0.9
LVDs (cm)	2.9 ± 0.7	3.2 ± 0.9	4.2 ± 1.3
EF (%)	58 ± 9	57 ± 8	39 ± 18
RVEDP (mm Hg)	19 ± 4	18 ± 5	NA
LVEDP (mm Hg)	17 ± 4	21 ± 6	NA
PCWP (mm Hg)	22 ± 4	19 ± 4	NA

Data are expressed as mean value ± SD.
*$p < 0.01$ vs. SR-CP.
AF = atrial fibrillation; CP = constrictive pericarditis; EF = left ventricular ejection fraction; LAD = maximal left atrial dimension; LVDd = left ventricular end-diastolic dimension; LVDs = left ventricular end-systolic dimension; LVEDP = left ventricular end-diastolic pressure; NA = not applicable; PCWP = pulmonary capillary wedge pressure; RVEDP = right ventricular end-diastolic pressure; SR = normal sinus rhythm.

electrical rhythm and the control group with AF without CP. The left atrial size was significantly greater in patients with CP and AF than it was in patients with CP and sinus rhythm; however, the left ventricular size and systolic function were normal in both groups of patients with CP. All of the patients with CP showed elevated pulmonary capillary wedge pressure and equally elevated right and left ventricular end-diastolic pressures.

Respiratory variation in patients with CP and sinus rhythm. The MV-E velocity and E-VTI significantly decreased from expiration to inspiration (Table 2). The MV-A velocity significantly decreased from expiration to inspiration, whereas there was no change in A-VTI. As a result, the E/A tended to decrease and the E/A-VTI significantly decreased from expiration to inspiration. The E-wave deceleration time did not show respiratory variation.

Table 2. Respiratory Change in Patients With CP and Normal Sinus Rhythm

	Inspiration	Expiration	%E (%)
Mitral inflow			
Peak velocities			
Peak E (cm/s)	61 ± 17‡	75 ± 20	18.4 ± 11.5
Peak A (cm/s)	42 ± 14†	49 ± 18	11.8 ± 16.8
E/A	1.54 ± 0.58	1.64 ± 0.48	3.4 ± 26.2
E-DT (ms)	112 ± 33	115 ± 33	0.8 ± 20.8
Velocity time integral			
E-VTI (cm)	7 ± 3‡	8 ± 3	21.1 ± 14.0
A-VTI (cm)	4 ± 2	5 ± 2	2.0 ± 37.0
E/A-VTI	1.67 ± 0.63*	2.00 ± 0.82	11.7 ± 27.4
Pulmonary venous flow			
Peak velocities			
Peak S (cm/s)	43 ± 14‡	50 ± 17	12.9 ± 10.3
Peak D (cm/s)	33 ± 15‡	45 ± 18	26.7 ± 15.4
S/D	1.61 ± 1.03†	1.27 ± 0.72	−23.9 ± 30.0
Velocity time integral			
S-VTI (cm)	8 ± 4†	9 ± 4	13.5 ± 19.1
D-VTI (cm)	4 ± 3‡	7 ± 3	37.5 ± 22.4
S/D-VTI	2.74 ± 2.38‡	1.65 ± 1.19	−66.2 ± 35.7

Data are expressed as mean value ± SD. *$p < 0.05$; †$p < 0.001$; ‡$p < 0.0001$ vs. data during expiration.
A = mitral inflow late filling wave; CP = constrictive pericarditis; D = pulmonary venous diastolic wave; E = mitral inflow early filling wave; E-DT = deceleration time of early diastolic mitral inflow velocity; S = pulmonary venous systolic wave; VTI = velocity time integral; %E = respiratory variation.

Table 3. Respiratory Change in Patients With CP and AF

	Inspiration	Expiration	%E (%)
Mitral inflow			
Peak velocities			
Peak E (cm/s)	64 ± 20‡	76 ± 24	15.1 ± 4.5
E-DT (ms)	127 ± 44	118 ± 39	−9.1 ± 21.8
Velocity time integral			
E-VTI (cm)	8 ± 3†	10 ± 5	19.2 ± 7.9
Pulmonary venous flow			
Peak velocities			
Peak S (cm/s)	27 ± 6	33 ± 7	14.3 ± 28.6
Peak D (cm/s)	37 ± 8‡	58 ± 10	35.2 ± 12.2
S/D	0.77 ± 0.29*	0.59 ± 0.14	−35.4 ± 53.4
Velocity time integral			
S-VTI (cm)	5 ± 1	5 ± 2	10.6 ± 34.3
D-VTI (cm)	5 ± 2†	9 ± 4	44.6 ± 14.1
S/D-VTI	0.96 ± 0.53*	0.63 ± 0.27	−89.9 ± 139.9

Data are expressed as mean value ± SD. *$p < 0.05$; †$p < 0.001$; ‡$p < 0.0001$ vs. data during expiration.
AF = atrial fibrillation; other abbreviations as in Table 2.

Both the PV-S and D velocities as well as the S-VTI and D-VTI significantly decreased from expiration to inspiration. The %E in the D velocity and D-VTI was significantly greater than that in the S velocity and S-VTI ($p < 0.0001$ and $p < 0.001$, respectively) and that in the E velocity and E-VTI ($p < 0.01$ and $p < 0.001$, respectively). As a result, both the S/D and S/D-VTI significantly increased from expiration to inspiration. The %E in the S/D-VTI was significantly greater than that in the E/A-VTI ($p < 0.001$).

Respiratory variation in patients with CP and AF. The MV-E velocity and E-VTI significantly decreased from expiration to inspiration (Table 3). The PV-D velocity and D-VTI significantly decreased from expiration to inspiration, whereas the PV-S velocity and S-VTI did not change. As a result, the S/D and S/D-VTI significantly increased from expiration to inspiration.

The %E in the D velocity was significantly greater than that in the E velocity ($p < 0.001$). The %E in the D-VTI was significantly greater than that in the S-VTI ($p < 0.05$) and E-VTI ($p < 0.001$), respectively.

Respiratory variation in patients with AF without CP. There was no significant difference from expiration to inspiration in MV and PV velocity variables in patients with AF without CP (Table 4).

Comparison between sinus rhythm and AF in patients with CP. There was no significant difference in the %E of MV and PV velocity variables between sinus rhythm and AF in patients with CP (Table 5).

DISCUSSION

The marked respiratory variation in pulsed Doppler MV and PV velocities in patients with CP has been used to differentiate CP from restrictive cardiomyopathy (5–7). However, the difference in the respiratory variation between

Table 4. Respiratory Change in Patients With AF Without CP

	Inspiration	Expiration	%E (%)
Mitral inflow			
Peak velocities			
Peak E (cm/s)	88 ± 22	88 ± 21	−0.9 ± 1.6
E-DT (ms)	97 ± 23	96 ± 20	−0.8 ± 10.0
Velocity time integral			
E-VTI (cm)	11 ± 3	11 ± 3	−0.8 ± 5.7
Pulmonary venous flow			
Peak velocities			
Peak S (cm/s)	28 ± 28	28 ± 25	5.6 ± 16.5
Peak D (cm/s)	47 ± 10	46 ± 11	−2.1 ± 15.6
S/D	0.53 ± 0.47	0.57 ± 0.54	4.5 ± 10.4
Velocity time integral			
S-VTI (cm)	3 ± 2	3 ± 2	7.1 ± 15.9
D-VTI (cm)	6 ± 1	6 ± 2	−2.5 ± 17.7
S/D-VTI	0.47 ± 0.44	0.58 ± 0.63	5.5 ± 18.8

Data are expressed as mean value ± SD.
Abbreviations as in Table 3.

MV and PV velocities and the influence of AF on the respiratory variation was not well described. This study further demonstrates a significant respiratory variation both in MV and PV flow velocities with a greater rate of change in the PV velocities than in the MV velocities in patients with CP, including those with AF.

Respiratory variation in the normal subjects. It has been reported that the respiratory variation in the normal volunteers had a mean of less than 5% variation in MV-E velocity, with no subject showing a variation >15% (5). We have also reported that the normal subjects have shown very little respiratory variation in MV and PV velocities (4% and 7%, respectively) (15). Pulmonary veins, which are not surrounded by pericardium, are affected by intrathoracic pressure. In the normal subjects, change in the intrathoracic pressure is transmitted to cardiac chambers. Intrathoracic (pulmonary vein) and intracardiac (left ventricle) pressures similarly change with respiration. As a result, the MV and PV velocities in the normal subjects do not show significant respiratory variation.

Respiratory variation in patients with CP and sinus rhythm. We observed significant respiratory variation in both pulsed Doppler MV and PV velocity variables in patients with CP and sinus rhythm (Fig. 1). In patients with CP, the respiratory changes in intrathoracic pressure are not transmitted to the cardiac chambers because the encompassing thick pericardial scar separates intrathoracic pressures from intracardiac pressures (5). Moreover, interventricular dependence is exaggerated because of the limited constraints of the intrapericardial space (5,13,16,17). Those two factors caused accentuated respiratory variations of the MV and PV velocities. The change in the intrathoracic pressure caused greater respiratory variation of pressure in the pulmonary veins, which are not surrounded by pericardium, with little effect on pressures in the left ventricle, which is shielded by the thickened pericardium. Hence, the pressure gradient between the pulmonary vein and left ventricle, thereby the PV velocities, are reduced during inspiration (7).

Both the MV-E and PV-D waves are generated by the blood transportation from the pulmonary vein to the left ventricle as a left atrial conduit function (18). However, this study showed greater respiratory variation in the D velocity variables and S/D ratio than it did in the E velocity variables and E/A ratio, respectively. The mechanism of the difference could be explained in that the respiratory changes in the intrathoracic pressure were directly reflected on the respiratory change of pressure gradient between the PV and left atrium because of the proximity of these structures. Interestingly, we observed a significantly greater %E in the PV-D velocity in five of 31 patients with CP and sinus rhythm when their %E in the MV-E velocity <10% (19.6 ± 6.4 vs. 0.6 ± 7.2%, $p < 0.05$). In this study, there was a slight shortening in the E-wave deceleration time during inspiration similar to the results of Hatle et al. (5) and Oh et al. (17). The short deceleration time represents premature cessation of the left ventricular filling caused by constrictive physiology.

Influence of AF. We found similar respiratory variation in the MV-E and PV-D velocity variables, even in patients with CP and AF, with greater respiratory variation in the D velocity variables than in the E velocity variables. There was also greater %E in the PV-D velocity in one of 10 patients with CP and AF with %E in the MV-E velocity <10% (34.5% vs. 5.1%). Strikingly, eight of 10 patients with CP and AF with the %E in the E velocity <25% have shown %E in the D velocity ≥25%. This suggests that the evaluation of the respiratory variation in the PV flow, especially in patients with AF, could be more helpful in diagnosing CP than the MV inflow. The MV and PV velocities are usually affected by the irregular cardiac cycle lengths in AF (18,19). In the subjects with AF without CP, early diastolic MV and PV velocities become greater with longer filling intervals as shown in Figure 2 by white arrows (20). We could expect that, in patients with CP and AF, the filling interval should be a similarly important factor determining MV-E and PV-D velocities, even when the ventricular filling is limited to the first third of diastole. On the

Table 5. Comparison of the Respiratory Variation in Patients With CP and SR and With AF

	Mitral Inflow		Pulmonary Venous Flow					
%E	Peak E	E-VTI	Peak S	Peak D	S/D	S-VTI	D-VTI	S/D-VTI
SR	18.4 ± 11.5	21.1 ± 14.0	12.9 ± 10.3	26.7 ± 15.4	−23.9 ± 30.0	13.5 ± 19.1	37.5 ± 22.4	−66.2 ± 102.4
AF	15.1 ± 4.5	19.2 ± 7.9	14.3 ± 28.6	35.2 ± 12.2	−35.4 ± 53.4	10.6 ± 34.3	44.6 ± 14.1	−89.9 ± 139.9

Data are expressed as mean value ± SD. p = NS.
SR = normal sinus rhythm; other abbreviations as in Table 3.

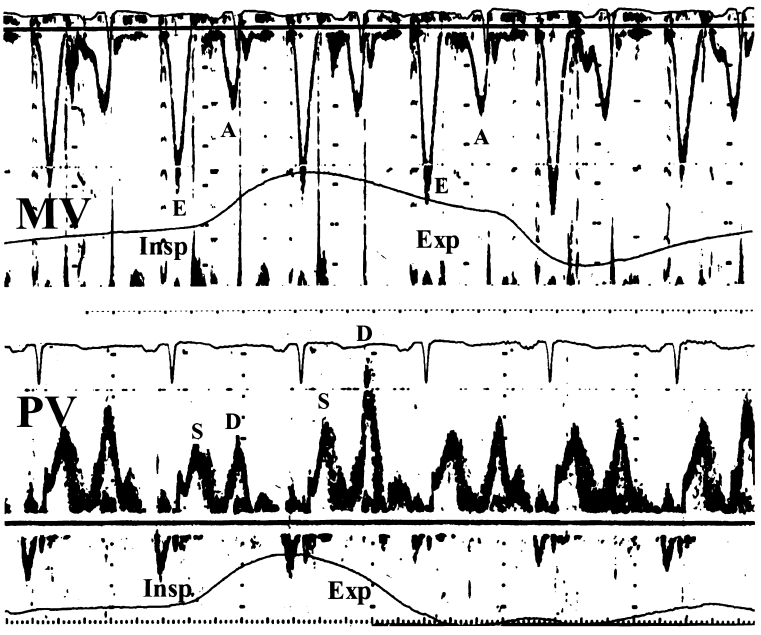

Figure 1. Respiratory variation in pulsed Doppler MV and PV velocities in patients with constrictive pericarditis and sinus rhythm. The MV-E velocity decreased from Exp to Insp. The MV-A velocity slightly decreased from Exp to Insp. Both of the PV-S and D velocities dramatically decreased from Exp to Insp. A = mitral inflow late filling wave; D = pulmonary venous diastolic wave; E = mitral inflow early filling wave; Exp = expiration; Insp = inspiration; MV = mitral inflow; PV = pulmonary venous flow; S = pulmonary venous systolic wave.

other hand, the constrictive physiology and hemodynamics could also be considered to influence the Doppler flow velocities in relation to the respiration. We observed a decrease in the E and D velocities during a long filling interval immediately after the onset of inspiration in patients with CP and AF as shown in Figure 3 by the thin arrows. This may be because the decrease in the pressure gradient during inspiration reduces blood volume from the pulmonary vein to left ventricle even with a longer cardiac cycle length. In contrast, we found an increase in those velocities during short filling interval at the onset of expiration as shown in Figure 3 by a thick arrow. The increased

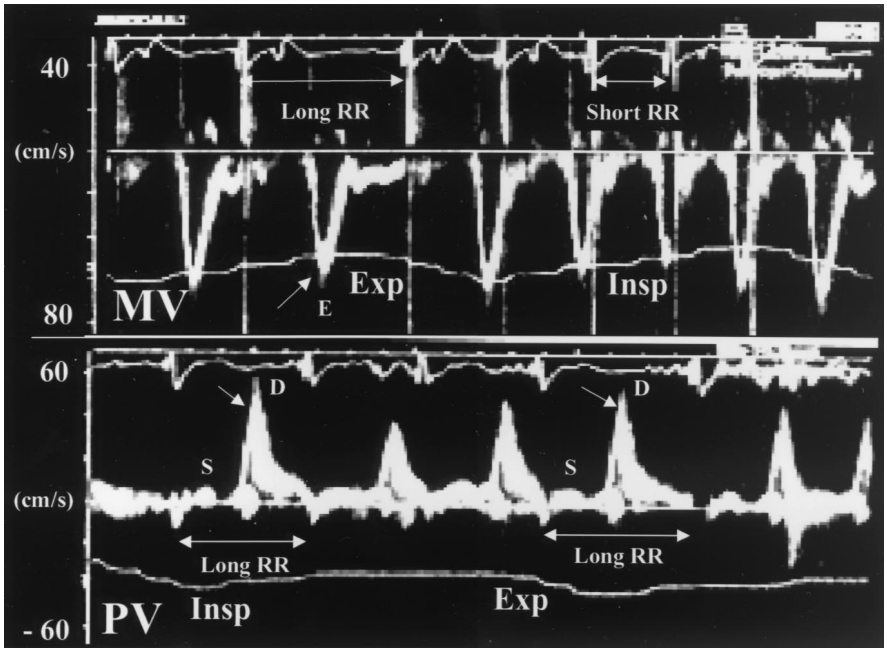

Figure 2. The beat-to-beat change in pulsed Doppler MV and PV velocities in patients with atrial fibrillation without constrictive pericarditis. In the subjects with atrial fibrillation without constrictive pericarditis, early diastolic MV and PV velocities become greater with long filling intervals as shown by **white arrows** regardless of the phase of respiration. D = pulmonary venous diastolic wave; E = mitral inflow early filling wave; Exp = expiration; Insp = inspiration; MV = mitral inflow; PV = pulmonary venous; S = pulmonary venous systolic wave.

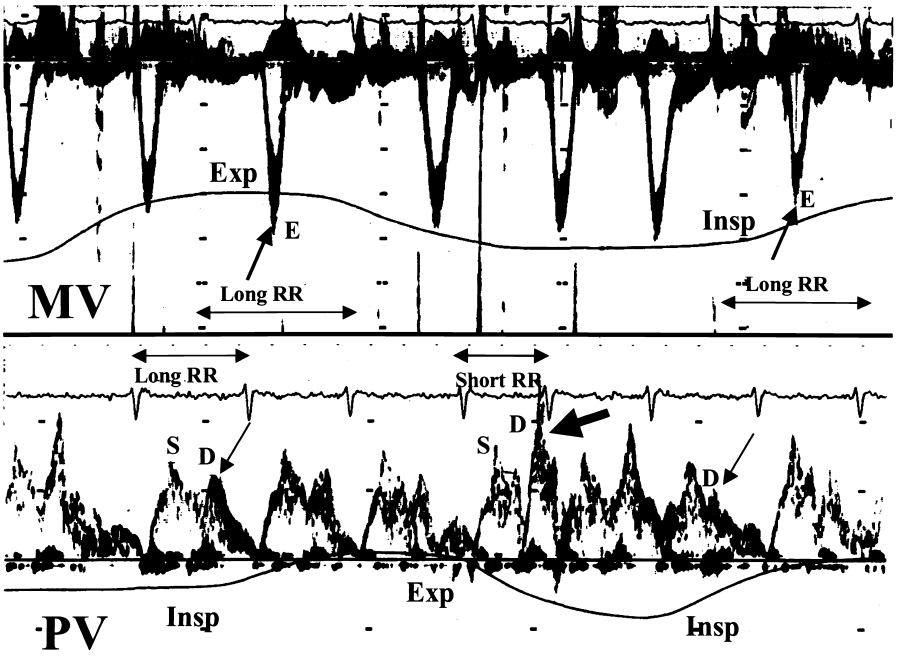

Figure 3. Respiratory variation in pulsed Doppler MV and PV velocities in patients with constrictive pericarditis and atrial fibrillation. A decrease in the MV-E and PV-D velocities during a long filling interval immediately after the onset of inspiration was observed as shown by the **thinner arrows**. In contrast, there was an increase in those velocities during short filling interval at the onset of expiration as shown by a **thick arrow**. D = pulmonary venous diastolic wave; E = mitral inflow early filling wave; Exp = expiration; Insp = inspiration; MV = mitral inflow; PV = pulmonary venous; S = pulmonary venous systolic wave.

pressure gradient between the pulmonary vein and left ventricle during expiration could transport enough blood volume, even with a shorter filling interval.

There was no significant respiratory variation observed in the PV-S velocity variables during AF. The S-wave is generated by the blood flow from the pulmonary vein to left atrium during ventricular systole as a reservoir function (21). It is affected by several factors, including left ventricular systolic function (22). During AF, the left ventricular systolic function varies beat-to-beat affected by preceding and prepreceding RR intervals with the mechanisms of postextrasystolic potentiation and mechanical restitution (19,23). Thus, the preceding cardiac cycle lengths might affect the PV-S velocity. It was reported that the S velocity in patients with AF with elevated left atrial pressure was lower than it was in patients with lone AF (23). The mean pulmonary capillary wedge pressure in patients with CP and AF in this study was greater than the normal range. For these reasons, the impact of respiratory variation on S velocity was lessened in AF.

Study limitations. Hatle et al. (5) described ≥25% expiratory increase in the MV-E velocity was characteristic in all patients with CP. Oh et al. (17) reported that 88% of the patients with CP have shown ≥25% expiratory increase in the MV-E velocity. In contrast, in this study, the average of the %E in the MV-E velocity was 18.4% in sinus rhythm and 15.1% in AF, which was statistically significant. Therefore, we did not regard the %E in the E velocity <25% as a nondiagnostic value for CP. It has been noted by Oh et al. (25) that, when the respiratory variation is masked in suspected CP, preload reduction by head-up tilt is recommended to unmask the respiratory variation. However, in our study, this maneuver was not performed systematically in all patients. We excluded patients with CP who had features of mixed constrictive and restrictive physiology and the patients with effusive CP in order to evaluate respiratory variation in patients with pure CP. Fifteen (37%) of 41 patients who did not undergo surgical treatment might have a lesser degree of CP. However, there was no significant difference in the %E in the PV diastolic flow between patients with and without pericardiectomy. Disappearance of the Doppler respiratory variation after pericardial stripping (5) and the use of the tissue Doppler echocardiography and M-mode color Doppler flow propagation velocity (26) could support our observations. The effect of varying cycle lengths on the Doppler parameters in AF could have influenced our findings. However, we did average 12 cardiac cycles in patients with AF. Temporal analysis might separate out the impact of variable cycle lengths and respiratory phase. In this study, we did not measure the respiratory variation of the right-sided Doppler flows because of the difficulty of obtaining these flows accurately using TEE. On the other hand, TEE could be useful in evaluating the localization and degree of pericardial thickening (27).

Clinical implications. Conventional M-mode and two-dimensional echocardiograms, computed tomography and magnetic resonance imaging have been regarded as an important noninvasive diagnostic tool for CP. However, not all of them reveal typical characteristics of CP. This study suggested that the evaluation of the respiratory variation in

the MV and PV velocities, regardless of the cardiac rhythm, might provide us additional information for diagnosing CP.

Conclusions. Significant respiratory variation in both MV and PV velocities occurs in patients with CP—not only in patients with normal sinus rhythm but also in those with AF. Moreover, the respiratory variation was greater in the PV velocities than it was in the MV velocities. Evaluation of the respiratory variation in the PV velocity variables using TEE can be a sensitive diagnostic strategy for evaluation of CP even in patients with AF.

Reprint requests and correspondence: Dr. Allan L. Klein, Department of Cardiology, Desk F-15, the Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44195. E-mail: kleina@ccf.org.

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